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SUBDURAL EFFUSIONS IN INFANCY: 24 CASES

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Subdural effusions in infancy have received little attention from British writers. Brief communications have been made by Northfield (1939, 1 case), Hinden (1939, 1 case), and Gimson (1945, 3 cases); while Wolfe (1948) has reported two cases of subdural haematoma treated successfully by operation, and Fine and Herson (1951) a fatal case. While this paper was in preparation Everley Jones (1952) recorded six cases of subdural effusions complicating purulent meningitis.

It should be emphasized that infantile subdural effusion is not a rare condition. Study of the records of the Royal Manchester Children's Hospital for the four years covered by this series shows that, of all surgical conditions of the central nervous system occurring in the first two years of life, only spina bifida and hydrocephalus were seen more often than subdural haematoma. Tumours and abscesses of the central nervous system in infants were much less common. Similarly, Smith and her co-workers (1951) have reported finding subdural effusions in almost a half of their cases of bacterial meningitis in infancy, and Everley Jones's (1952) figures are similar. To paraphrase Ingraham and Heyl (1939), the frequency with which these effusions are found is proportional to the intensity with which they are sought.

The series here reported comprises 24 cases—18 cases of subdural haematoma in infancy; 5 cases complicating purulent meningitis; and 1 case of subdural effusion in a patient with sagittal sinus thrombosis. These groups are considered separately.

SUBDURAL HAEMATOMA IN INFANCY

The recognition of the importance of this condition is virtually the work of one man—Franc Ingraham, of Boston, whose classical papers with Matson (1944, 1949) record a much larger series of cases than any other writer. A further 55 cases are reported by Elvidge and Jackson (1949). Their series includes 10 cases of birth trauma treated by immediate operation and 9 cases of severe post-natal head injury, both of which groups had a very high mortality—even in the more chronic group 19.2% died. Statten (1948) reported 13 cases treated by miscellaneous methods and compared them with 15 cases treated by the regime advised by Ingraham and Matson (1944). He showed that in the former group inadequate treatment led to many deaths and to mental deficiency among the survivors; in the latter, 10 cases were normal.

Age and Sex.—Most cases occur in the first few months of life. Of the series reported here 11 developed symptoms before the age of 3 months, five between 3 and 6 months, and only two thereafter (Table I). Ingraham and Matson (1949) found a preponderance of males over females in the ratio of 5:3. In this series the preponderance is even more striking, being 5:1.

Aetiology and Pathology

Ingraham and Matson (1949) found evidence of birth trauma in about a quarter of their cases. In the present series a comprehensive history was available in 16 cases. Eight of these sustained definite birth trauma with a history of obvious damage to the head, difficulty in resuscitation, or a bloody cerebrospinal fluid in the neonatal period. Two infants were members of twin pregnancies and two were born prematurely (at 30 and 34 weeks respectively). In a further two cases the baby had sustained a definite head injury a fortnight before the onset of symptoms, though no sign of fracture of the skull was found on *x*-ray examination. In only two cases was the antecedent history completely negative.

Obviously, therefore, subdural haematoma is very often associated with an abnormal or difficult labour (75% in this series). Furthermore, the fact that it most frequently presents in the first months of life also suggests that the cause is to be sought at or near the time of birth; finally, the preponderance of males might be explained on the basis that male infants have bigger heads than females, and are therefore more likely to sustain intracranial injury at birth.

There is no evidence that the haemorrhagic diseases play much part in the aetiology of the condition. Ingraham and Matson have noted that 31.5% of their cases showed some anaemia, but this is a common finding in infants who have been ill for long, whatever the cause. In the present series haemoglobin estimations were made at the beginning of treatment in 14. Nine of these infants had been ill for 14 days or less; only two of them showed some anaemia, while in seven the haemoglobin was within the normal limits for age. Of the five infants who had been ill for more than 14 days three showed a haemoglobin of less than 60%; in the other two it was 68%.

Much has been written on the pathology of subdural haematoma. The conventional theory is that the original haemorrhage occurs from a cortical vein, probably in many cases one crossing the subdural space to reach a major venous sinus. This haemorrhage clots, and the clot then liquefies and eventually becomes surrounded

TABLE I.—*Clinical Features in 18 Cases of Infantile Subdural Haematoma*

Case No.	Sex	Age at Onset of Symptoms	Duration of Symptoms	Obstetric Abnormality	Presenting Symptoms	Enlargement of Head	Site of Haematoma	Craniotomy	Result
1	F	2 months	14 days	Birth injury	Fits, enlarged head	1 in.	L. and R.	L. and R.	Developing normally (4 yrs.)
2	M	3 ,,	1 day	Not known	Fits	1½ ,,	,,	,,	Operative death (2nd craniotomy)
3	M	2 ,,	3 days	Twin	Fits, vomiting	1½ ,,	,,	,,	Well for 1 year. Untraced recently
4	M	2 ,,	14 ,,	Birth injury	Vomiting, enlarged head	1½ ,,	,,	,,	Developing normally (3 yrs.)
5	M	10 ,,	7 ,,	Premature	Fits	No	,,	No	Died in convulsions within 1 year
6	M	5 ,,	8 ,,	*Nil.* Head injury 2 weeks before admission	Vomiting	1 in.	,,	L. and R.	Developing normally (3 yrs.)
7	M	2 ,,	4 months	Premature	Fits and left hemiparesis	1½ ,,	,,	,,	Backward (2 years); left hemiparesis
8	M	Newborn	14 days	Birth injury	,, ,, ,,	No	R.	R.	Mentally normal (2 years); left hemiparesis
9	M	3 months	1 month	,, ,,	Fits	,,	R. (small)	No	Unimproved. Died of convulsions
10	M	3 ,,	4 days	,, ,,	Fits and vomiting	,,	L. and R.	L. and R.	Developing normally (18 months)
11	M	4 ,,	6 weeks	,, ,,	Fits	,,	L. (small)	No	Unimproved. Has cerebral palsy
12	F	1 month	14 days	*Nil*	Vomiting, screaming attacks	1½ in.	L. and R.	L.	Developing normally (18 months)
13	M	12 months	10 ,,	*Nil.* Head injury 2 weeks before admission	Vomiting and enlarged head	1 ,,	,,	L. and R.	Developing normally (1 year)
14	M	2 weeks	14 ,,	Precipitate labour	Fits and bulging fontanelle	No	,,	No	Developing normally (6 months)
15	F	3 ,,	5 weeks	Twin	Fits and vomiting	,,	,,	,,	Appears well (1 month)
16	M	2 months	4 ,,	*Nil*	Vomiting and one fit	,,	,,	L. and R.	,, ,, ,, ,,
17	M	1 month	13 months	Birth injury	Fits and backwardness	,,	L.	L.	Unimproved, membrane irremovable
18	M	2 months	7 years	Not known	Enlarged head, vomiting, headache	4 in.	L. and R.	L. and R.	Well at present (3 months)

by a semipermeable membrane of fibrous tissue. The liquefied clot has a high protein content and therefore attracts fluid into itself by osmosis. Thus it continues to expand for a long period after its original formation, though if a fatal issue does not supervene the fluid may later gradually be absorbed and the walls either calcify or form a dense "scar" adherent externally to the dura and internally to the arachnoid and brain substance itself.

Symptoms and Signs

The commonest symptoms of infantile subdural haematoma are, in order of frequency, convulsions, vomiting, and irritability. Occasionally the parents notice enlargement of the head. On examination more than half the cases were found to have some pyrexia and unduly brisk tendon reflexes. More than half showed either a significant degree of enlargement of the head (that is, a circumference greater by 1 in. (2.5 cm.) or more than the normal for age) or a bulging fontanelle. It must be remembered that severe vomiting in an infant will rapidly cause dehydration, and there may therefore be the combination of enlarged head and depressed fontanelle as happened in Case 1. Changes in the optic fundi are rather uncommon (20–25%), the abnormality noted being much more often retinal haemorrhages than papilloedema.

Focal neurological signs are exceptional, but this is not wholly surprising, because chronic subdural haematoma in adults frequently gives rise to few or to very misleading neurological signs, and the more yielding nature of the baby's skull might be expected to lessen the local effects of compression still more. On the two occasions when a hemiplegia was observed before operation it failed to improve thereafter. It would seem, therefore, that focal signs may indicate underlying contusion or laceration of the brain rather than compression by the haematoma.

In only one case (No. 8) was there a complete third-nerve palsy, with ptosis and a fixed dilated pupil, on the side of the clot. This was a case of known birth injury with the immediate onset of convulsions. Operation cured the paralysis, which was doubtless due to compression of the nerve by herniation of a cerebral hemisphere through the tentorium. A further two cases had a single sixth-nerve palsy, as of course may happen in any case of increased intracranial pressure. Both cleared up rapidly during treatment.

Diagnosis

There is only one diagnostic procedure of value—the subdural tap. The technique is described in detail by Ingraham and Matson: briefly, a needle is inserted just through the dura at the lateral angle of the anterior fontanelle, and the fluid, if any, aspirated. Full aseptic precautions are necessary and a generous area of the scalp should be shaved. Not more than 10 ml. of fluid should be taken off from either side at the first tap, because the sudden removal of large quantities of fluid may result in collapse, hyperpyrexia, or renewed convulsions. It is unnecessary and unsafe to advance the needle into the brain—a blood vessel may be injured. If any quantity of subdural fluid is present it will be easily found, but where a strong suspicion of subdural effusion exists and the initial tap is negative a further tap may properly be done some days later. The original clot of blood may be relatively solid and circumscribed, but it seems to spread widely over the cortex as it liquefies.

Almost always the fluid obtained is xanthochromic, with a variable quantity of fresh blood; rarely it may be colourless but of a high protein content. In this series the protein content of the fluid varied from 1 to 5 g., with an average of 2 g. per 100 ml. There was an excess of white cells, the counts varying from 60 to 1,600 per c.mm. The polymorph/lymphocyte ratio was usually about 1:2.

There is little likelihood of confusing subdural fluid with cerebrospinal fluid. The latter was obtained by lumbar puncture in nine cases. In five it was of normal chemical composition, in two there was faint xanthochromia with a few red cells but a normal protein content, and in two the proteins were moderately elevated (100 and 170 mg. per 100 ml.). Moreover, it is almost impossible to extract more than a few drops of cerebrospinal fluid from the subarachnoid space over the surface of the brain by needling.

Air encephalography is unsafe in these, as in almost all other cases of raised intracranial pressure, and is best avoided. Angiography might be safe, but the diagnosis can be made without it.

Treatment

It is not proposed to give full surgical details of treatment. After the first finding of subdural fluid the taps are repeated daily, the amount extracted being gradually

increased to as much as 25 ml. from each side when the effusion is large. There is usually a dramatic improvement in the infant's condition within a matter of hours or at most a day or two. Convulsions and vomiting cease, the infant takes feeds readily and begins to gain weight again. The fontanelle becomes less tense and the head circumference may even shrink to normal.

By the tenth to the fourteenth day the infant is fit for operation. This consists, in the first instance, in making burr-holes in the temporal region on the side of the haematoma. When the clot is bilateral the burr-holes are made on both sides. The dura mater is opened and the subdural space inspected. In some cases—unfortunately the minority—a small amount of residual subdural fluid escapes and the brain re-expands at once; there is no membrane formation and the wounds can be closed. It is our practice to perform subdural taps once or twice during the next week. Usually no further fluid is found, and the child can be discharged from hospital.

In the other cases, comprising three-quarters of the total, one sees on opening the dura mater that a membrane already surrounds the haematoma. The outer layer of the membrane is adherent to the dura: it is the thicker of the two layers, and its inner surface is often shaggy. The inner layer is usually thin but tough, and adherent to the arachnoid.

The whole point of the method of treatment advocated by Ingraham is that this membrane must be removed if the important growth of the brain which occurs during the first two years of childhood is not to be arrested by its constricting influence. Accordingly, an osteoplastic flap is turned down and as much of the membrane as possible is dissected away from the surface of the hemisphere. Where the membrane is bilateral a flap is turned down on one side at a time. Recovery is usually so rapid that only 10 to 14 days need elapse between the two craniotomies. A blood transfusion is always given during operation, and is repeated if necessary a few days later. Occasionally there is some effusion into the subdural space during the early post-operative period. This is treated by repeated aspiration as described above and soon settles down. A recurrent effusion was only once seen in this series (Case 6). Some of the clinical features are shown in Table I.

Results

Ingraham and Matson have claimed that 70% of their cases developed into normal children. It is not surprising that the results fall short of perfection when one considers that many of these children are known to have sustained major intracranial injuries at birth, and when sometimes at operation one finds the cortex of the brain shrunken, yellow, and sclerotic or full of areas of cystic degeneration. What is more surprising is that the mortality is so low. Ingraham and Matson have had 5 deaths in 116 patients who were subjected to craniotomy. As the membranes were bilateral in exactly half their series, this represents 5 deaths in 174 operations. In the present series, Case 17, which is mentioned again later, had been neglected and his condition was in effect inoperable when he was first seen. In Case 5 craniotomy was advised but refused. It is a striking vindication of Ingraham's views that this child continued to have frequent convulsions and died in status epilepticus within a year.

This leaves 16 cases in which treatment was carried through according to plan, craniotomy being necessary in 12, including 10 bilateral cases. The solitary death (Case 2) was due to an error of judgment in failing to order an immediate blood transfusion. Of the 15 survivors, 8 are developing normally, the eldest being now at school, and 2 more are well, though their follow-up is too short to have significance. In Case 18 a diagnosis was made only after his subdural haematomas had been present for years. His head was greatly enlarged, and a bilateral removal of the subdural membranes was not followed by complete re-expansion of the brain. He is clinically well, with a normal intracranial pressure, and shows little mental retardation and no focal neurological signs.

There is reasonable cause, therefore, to hope that 10 or possibly 11 of the 16 patients will grow up into normal children, a figure which is comparable to that given by Ingraham and Matson. One patient died, and, of the other four, two (Cases 7 and 8) have residual neurological signs. These are the two who had a hemiparesis which was unrelieved by surgery; evidently their brains were severely damaged. There is some doubt whether in the final two cases (Cases 9 and 11) the subdural effusion had any relevance to their symptoms. In both its volume was very small, the total amount found over a period of 10 days being only 15 ml. and 25 ml. respectively, compared with a minimum of 100 ml. in any of the others. Neither showed membrane formation and neither was improved by subdural taps, though such fluid as was present was typically xanthochromic and had a vastly higher protein content than the cerebrospinal fluid examined at the same time. One now has cerebral palsy, the other died in convulsions.

Case 14. Subdural Haematoma without Membrane Formation

This patient was a male infant aged 2 weeks whose birth was precipitate. At the age of 2 days he developed convulsions and was admitted to a hospital, where lumbar puncture was performed. The fluid contained 170 mg. of protein per 100 ml. and 19 lymphocytes per c.mm. A diagnosis of subdural haematoma was made and he was transferred to the Royal Manchester Children's Hospital.

On admission he was found to have a head circumference of 14½ in. (37 cm.); the fontanelle was tense and bulging. His temperature was 99.4° F. (37.4° C.). A blood count showed: Hb, 68%; white cells, 10,000 per c.mm., differential count normal. Subdural taps yielded 5 ml. of turbid mahogany-coloured fluid, containing large numbers of blood cells, on each side. Subdural taps were made on each of the five subsequent days, a total of 100 ml. being withdrawn. By this time the child was so well that it was possible to make exploratory burr-holes.

Operation.—Under general anaesthesia temporal burr-holes were made on each side. On the left only a few millilitres of subdural fluid remained and there was no trace of membrane. On the right side some 60–80 ml. of fluid was evacuated, the brain expanding rapidly and completely. There was possibly a trace of roughening of the inner surface of the dura, as though a membrane were about to form, but the surface of the arachnoid was normal. The wounds were therefore closed.

Six days later a final subdural tap was made but no further fluid was found. The patient's convalescence was complicated by an attack of acute bronchitis, but he is now developing normally.

Case 6. Recurrent Subdural Haematoma with Membranes

A male baby of 5 months was admitted on February 7, 1949, with a history of having fallen out of his cot and banged his head on the floor two weeks before. He had vomited repeatedly for five days. On examination he was found to be very irritable and pale. The head circumference was 18½ in. (47 cm.), the fontanelle tense. There was a retinal haemorrhage in the right fundus. The tendon reflexes were grossly increased.

Subdural taps yielded 7 ml. of blood-stained xanthochromic fluid on each side, having a protein content of 1,500 mg. per 100 ml., 220,000 red cells, and 900 white cells. A further 60 ml. of fluid was obtained during the next four days, but thereafter taps were dry on two successive days and bitemporal burr-holes made under general anaesthesia showed no further effusion. The child was therefore discharged from hospital on March 2.

Some 12 weeks later (May 25) he was readmitted with a history of renewed vomiting with drowsiness, fever, and

cough for eight days. On examination the head circumference was as before, the fontanelle tense. His temperature was 101° F. (38.3° C.) and there was slight meningism. An immediate subdural tap yielded 8 ml. of very deeply blood-stained fluid on each side. The subdural taps were repeated daily for a further 18 days, 850 ml. of fluid being removed. The composition of this was similar in all respects, at first, to that obtained before. Later the fluid became almost clear yellow.

First Operation.—On June 13, under general anaesthesia and with a blood transfusion from the beginning, a right temporal osteoplastic flap was turned down. There was found to be a large cavity lined with a thin membrane in the subdural space extending mainly anteriorly over the frontal lobe of the brain. Most of the membrane was dissected away, the cavity filled with saline, and the wound closed. Convalescence was uneventful except for a considerable recurrence of effusion on the operated side during the next 14 days, a total of 210 ml. being aspirated.

Second Operation.—On June 27, again under general anaesthesia and with continuous blood transfusion, a left temporal osteoplastic flap was turned down. This time the haematoma was found to be mainly over the post-frontal, parietal, and temporal regions, and was easily dissected away.

The child again made a smooth recovery, and this time further aspirations of the subdural spaces were negative. He left hospital on July 20. He has since been seen at intervals of a few months in the out-patient department and is developing normally in all respects.

Case 17. Neglected Subdural Haematoma

This baby was admitted at the age of 14 months. His had been a very difficult forceps delivery, and he sustained a depressed fracture of the left side of the skull. At 4 days he had several fits (right side). At 10 weeks he had a febrile illness with screaming attacks and vomiting which lasted a week. Since then he had had repeated generalized epileptic attacks, and occasional projectile vomiting. He had not sat up or begun to play with toys.

On examination he was found to be very backward, paying no attention to sounds or to objects moved in front of him and making no effort to sit up. There was a gross depression of the left fronto-parietal region of the skull; the head circumference was normal. The pupils reacted briskly to light, the fundi were normal, and there was no obvious hemiparesis. Lumbar puncture showed 5 white blood cells per c.mm. with a normal chemistry.

First Operation.—Under general anaesthesia an exploratory burr-hole was made over the site of the depressed fracture, but nothing abnormal was found. A burr-hole over the left parietal eminence, however, revealed a rather tough subdural membrane, densely adherent to dura and brain, from which a small piece was removed for biopsy. It showed mature fibrous tissue with no evidence of calcification.

Second Operation.—A week later, under general anaesthesia, a small bone flap was turned down over the site of the biopsy. The dura was lined with a tough fibrous tissue several millimetres thick, adherent everywhere to the arachnoid. The subarachnoid space was abnormally deep, the cerebral cortex yellow and tough, with small atrophic gyri and wide sulci. In one area it contained multiple small cysts. There seemed no point in dissecting away the subdural membrane in the presence of such severe brain atrophy, so the bone flap was replaced in the usual way.

This child has not been seen recently, but was making little progress during the few months after his operation.

Comment

Case 14 shows how with early diagnosis one can hope to achieve a rapid and smooth recovery without recourse to major surgery. If the subdural spaces can be emptied early enough no membrane is allowed to form and there need be no craniotomy.

Case 6 taught a lesson which has since been emphasized by Ingraham and Matson (1949). The majority of these haematomas extend into the temporal region and will be found by the diagnostic burr-holes already recommended. From time to time, however, a case occurs in which the clot is situated elsewhere, so that, if the suspicion of subdural haematoma is very strong but temporal exploration is negative, further burr-holes should be made in the frontal and parietal regions. The sudden "drying-up" of the subdural spaces should have suggested that there was still some effusion, but in a situation not accessible to needling through the fontanelle. The recurrence of symptoms 12 weeks after the first admission was doubtless due to the further expansion of a haematoma which had been only partially aspirated before. It is fortunate that the delay has had no permanent effect on the child's mental development.

It would be wrong to claim that Case 17 could necessarily have been saved by prompt treatment. Although there was certainly an old subdural haematoma, the atrophy and sclerosis of the underlying cortex may well have been the end-result of contusion, laceration, and vascular disturbance occurring during the actual birth trauma.

SUBDURAL EFFUSION IN PURULENT MENINGITIS

There are several different circumstances in which infection and subdural effusion can be associated. Spitz *et al.* (1945) pointed out the fundamental distinction between subdural effusion complicating purulent meningitis and a collection of pus in the subdural space complicating osteomyelitis of the skull. Schiller *et al.* (1948) noted that the latter condition might be either localized or spreading. If it were spreading they preferred to call it "purulent pachymeningitis." They also mentioned the possibility of subdural abscess occurring as a complication of cerebral abscess or through the infection of a subdural haematoma. Finally, as is reported in the next section, a subdural effusion may complicate cerebral thrombophlebitis.

The complete classification of subdural effusions associated with infection is therefore :

- A. Localized
  - (1) With osteomyelitis of the skull.
  - (2) With cerebral abscess.
- B. Spreading
  - (1) With osteomyelitis of the skull—"purulent pachymeningitis."
  - (2) Due to infection of a subdural haematoma.
  - (3) With purulent meningitis.
  - (4) With cerebral thrombophlebitis.

Of the spreading effusions, purulent pachymeningitis has a strong tendency to loculate, and therefore presents a surgical problem of great difficulty. We are here concerned only with the meningitic variety, the five examples being shown in Table II.

TABLE II.—*Clinical Features in Five Cases of Subdural Effusion Complicating Purulent Meningitis*

Case No.	Sex	Age on Admission	Infecting Organism	Presenting Symptoms	Time from Onset of Meningitis	Site of Effusion	Craniotomy	Result
19	F	4 months	Not identified	Fits	4 weeks	L. and R.	L. and R.	Fits continued: died a year later
20	M	12 ,,	Streptococcus	Vomiting	1 week	,,	No	Developing normally (2½ years)
21	M	8 ,,	Pneumococcus	Fits	3 weeks	,,	L.	Behaviour disturbed (2 years)
22	F	6 ,,	*Staph. pyogenes*	,,	2 ,,	,,	No	Appears well (2 months)
23	F	12 years	Pneumococcus	Fits. Right hemiparesis	5 years	L.	L.	,, ,, (6 ,,)

Incidence.—Most of these cases occur in the first year of life (McKay *et al.*, 1950; Smith *et al.*, 1951; Everley Jones, 1952), but cases may be found in adults (Spitz *et al.*, 1945). The incidence between the sexes seems to be about equal, in contrast to the marked preponderance of males in subdural haematoma.

Aetiology and Pathology

The causative organism of the initial meningitis is most commonly *Haemophilus influenzae* or a pneumococcus, but several others have been recorded. In this series two were pneumococcal, one was staphylococcal, one streptococcal; and in one no organism was isolated. The reason for the effusion is obscure. McKay *et al.* mention the possibilities of exudation or haemorrhage into the subdural space as a complication of either antibiotic therapy or of repeated lumbar puncture, or of "minimal trauma in a critically ill infant"; Smith *et al.* postulate a transudation of fluid containing cells and bacteria across the inflamed arachnoid into the subdural space, where the lysis of the cells produces a fluid of high protein content, and osmotic processes subsequently cause the effusion to enlarge until it produces symptoms. It is known that an effusion can occur without intrathecal administration of antibiotics, while the fact that it does not complicate tuberculous meningitis must surely nowadays exclude lumbar puncture as a cause. There may in fact be two separate conditions. The minimum protein content in the nine cases of McKay *et al.* was 700 mg. per 100 ml. The 20 cases of Smith *et al.*, however, showed a variation between 40 and 1,920 mg. of protein per 100 ml. In Everley Jones's series there were four cases with a protein content of 1,000 mg. or more, and two in which it lay between 150 and 250 mg. per 100 ml. In the four patients now reported in whom the estimation was made, the reading was between 1,000 and 5,000 mg. per 100 ml.

It is tempting to suggest that the lower-protein group represent cases of simple transudation across the arachnoid, while in the high-protein group some additional element exists. A suggestion about the nature of this additional factor is made below. The difference is not due simply to the age of the effusion, for a high reading was found by Everley Jones in his Case 6 on the eighth day of the illness, and a low one in his Case 4 on the sixteenth day.

Whatever the reason, it is certainly unexpected and interesting to find in the present series that the protein content of the fluid is exactly the same as it is in subdural haematoma (1–5 g., average 2 g., per 100 ml.), nor were the white cell counts very dissimilar (30–2,500, usually 400–600, per c.mm.).

Diagnosis and Treatment

It is generally agreed that a subdural effusion should be suspected, and diagnostic subdural taps made, in any infant with meningitis whose clinical progress is unsatisfactory after three days' treatment, or who has persistent fever, vomiting, convulsions, or a tense fontanelle.

The routine of daily subdural taps, exactly as advised for subdural haematoma, should be followed. After 10 days, at any rate in all but the smallest effusions, bitemporal burr-holes should be made and the subdural space inspected. Where membrane formation has occurred, craniotomy with removal of the membrane is indicated.

In Case 20 the fontanelle had closed and the diagnosis was made by exploratory burr-holes. Here membrane formation was minimal and a good result was obtained by simple tube drainage for four days.

Results

No one yet knows the long-term results in these cases. In Case 23 (this patient is older than the others, but is included here because her condition was the same as that of the infants) the membrane was too adherent and widespread to be totally removed. Of the two cases in which only a very thin membrane was present, Case 20 has been followed for 2½ years and is developing normally, and Case 22 seems well but not enough time has elapsed for assessment. Of the two with membranes requiring craniotomy, Case 19 was always backward, suffered from fits, and died a year after operation from a further attack of meningitis. Case 21 has been followed for two years, and is a bad-tempered and intractable child.

Nevertheless, from the literature referred to previously one gets the general impression that with early diagnosis the results may approach those obtained in infantile subdural haematoma.

Case 20. Effusion without Membrane Formation

A male baby aged 12 months was transferred to the Royal Manchester Children's Hospital with a seven-day history of streptococcal meningitis complicating a left otitis media. He was pyrexial and vomited repeatedly. There was a left external rectus palsy, the head circumference was 20¼ in. (51.5 cm.), and *x*-ray films of the skull showed gross separation of the sutures. There were no localizing neurological signs. The lumbar cerebrospinal fluid contained 110 mg. of protein per 100 ml. and 33 cells per c.mm. Bitemporal burr-holes made under general anaesthesia revealed large collections of turbid yellow subdural fluid. This contained 3.75 g. of protein per 100 ml. and 2,500 white blood cells (80% polymorphs). As there was only a very thin membrane craniotomy was not performed. Instead the subdural spaces were drained by rubber tubes for four days. The child made an uninterrupted recovery, and aspiration of the burr-holes seven days after the removal of the tubes showed no evidence of recurrence of the effusion. As noted above, he is developing normally.

Case 21. Subdural Effusion with Membranes

A male infant of 8 months was admitted to the Royal Manchester Children's Hospital on March 13, 1950, with a history of vomiting and a squint for three days, followed by convulsions within the last 24 hours. On examination he was having frequent right-sided epileptic attacks, there was no meningism, and the fundi were normal. There were rales over both lung fields. Immediate lumbar puncture yielded a turbid fluid containing 500 white cells per c.mm. and many Gram-positive diplococci, later established as pneumococci: the protein content was 300 mg. per 100 ml. Treatment with penicillin intramuscularly and intrathecally together with sulphadimidine was begun at once. By March 30 the cerebrospinal fluid showed 45 cells (all monocytes) per c.mm., 110 mg. of protein per 100 ml., and sterile culture, but the child was still irritable and pyrexial, and was developing a right hemiparesis. As the fontanelle was closed and the diagnosis was uncertain, bilateral occipital burr-holes were made on March 30 with a view to ventriculography if necessary. These showed the presence of large subdural effusions on each side, the fluid being xanthochromic and slightly turbid, with a protein content of 5,000 mg. per 100 ml. The wounds were closed and a further 80 ml. of similar fluid was aspirated during the next week from the left side. The right side produced only a further 20 ml. of fluid.

Craniotomy.—On April 10, under general anaesthesia, a left temporal osteoplastic flap was turned down and a rather thin but tough subdural membrane dissected away. The cavity was filled with saline and the flap replaced and sutured.

During the immediate post-operative period there was some pyrexia and swelling of the scalp, but no frank sepsis developed. Further aspirations of the left subdural space were negative. On May 1 a right temporal burr-hole was made, but there were no signs of effusion or membrane formation on this side. Meanwhile, the right hemiparesis had almost completely cleared, and the child was discharged a week later. Two years later he was bad-tempered, destructive, and dirty in his habits. There was still slight stiffness of the right arm and leg.

Case 23. Neglected Subdural Effusion

A girl aged 12 years was admitted some five years before with a history of pneumococcal meningitis which had been complicated by the development of a right hemiparesis, dysphasia, and fits. Cerebral thrombophlebitis had been suspected at the time. When seen at the Royal Manchester Children's Hospital she was having about one major and one minor epileptic fit per week, the right face and limbs twitching more than the left. She had some difficulty in speech and definite clumsiness of the right hand, but a normal gait. Air encephalography showed a gross dilatation of the posterior part of the left lateral ventricle.

Operation.—Under general anaesthesia a left-sided posterior osteoplastic flap was turned down. The dura was found to be everywhere densely adherent to a membrane about 1 cm. in thickness. This in turn was very adherent to the underlying cortex, and only a limited amount could be dissected away. There was also, on the other hand, a well-localized cerebral scar; this was excised down to the ventricle, leaving healthy white matter on all sides. The usual closure of the wound was made.

The child made a good recovery. There has been no increase in her neurological signs and so far (six months after operation) she has had no further fits.

Comment

In Case 21 the relatively poor result may be due either to the severity of the original infection or to the fact that diagnosis was somewhat delayed. In Case 23, which closely resembles the neglected case of subdural haematoma (Case 17) there was so typical a sheet of fibrous tissue lying in the subdural space and extending far beyond the bounds of the cerebral scar that the diagnosis of old encapsulated subdural effusion can hardly be doubted. It is conceivable, however, that the findings represent spontaneous resolution of a cerebral abscess with a subdural effusion overlying it.

The high proportion of cases with membrane formation recorded by McKay *et al.* (1950) and in this series, both from neurosurgical clinics, contrasts with the very much lower figures of cases submitted to operation from paediatric clinics: Smith *et al.* (1951)—two cases in twenty—and Everley Jones (1952), one in six. The explanation is doubtless that if subdural effusions are sought for early in cases of meningitis they will rapidly respond to treatment, without need for major neurosurgical intervention. But where they are not so sought, though a proportion may resolve spontaneously, the others will become encapsulated. These latter, if untreated, may cause either a fatal issue or (as in Case 23) serious disability later.

CEREBRAL VENOUS THROMBOSIS WITH SUBDURAL EFFUSION

The whole question of the importance of cerebral thrombophlebitis as a cause of neurological symptoms and signs in infancy is a somewhat controversial one. The condition is rarely fatal, and even modern cerebral angiography with special methods designed to demonstrate the venous sinuses has failed, both in cases investigated by me and in those of others (Logue, 1952, personal communication), to demonstrate an obstruction with any regularity. None the less there are certain forms of illness in infants, particularly with a sudden onset of fits and a varying degree of hemiplegia associated with fever and moderate increase in the cells and protein content of the cerebrospinal fluid, in which the diagnosis of cerebral thrombophlebitis provides an acceptable explanation. The occurrence of a subdural effusion in such a case has some theoretical importance.

Case 24.—A female infant aged 4 months was admitted to Booth Hall Hospital under the care of Dr. W. H. Patterson, by whose kindness I record this case. She was the daughter of an epileptic mother and suffered from congenital heart disease. The immediate reason for admission was a history of five convulsions in the past ten days, with irritability and vomiting. On examination the child was severely dehydrated, but with a tense bulging fontanelle. Lumbar puncture showed a faintly xanthochromic fluid under a pressure of more than 300 mm. It contained 40 mg. of protein per 100 ml. and 12 leucocytes, 50% being lymphocytes and 50% endothelial cells. Subdural taps yielded 5 ml. on the left side and 3 ml. on the right of a dark-brown slightly blood-stained fluid containing 110 leucocytes on the left and 60 leucocytes on the right. The child was not improved by this procedure, and died within 48 hours. Necropsy showed a thrombosis of the sagittal sinus and of its tributary veins on both sides, together with pulmonary stenosis and a patent interventricular septum.

Comment

The similarity, already noted, of the fluid found in cases of infantile subdural haematoma to that found in the majority of cases of subdural effusion complicating purulent meningitis suggests that a similar cause is responsible for both. The haematomas are generally believed to arise from haemorrhage from a vein bridging the subdural space from cortex to venous sinus, so that it would seem reasonable to suppose that a similar effusion in purulent meningitis might arise from necrosis of the wall of a vein involved in a process of thrombophlebitis. Spitz *et al.* (1945) have already made this suggestion, claiming indeed to demonstrate in their necropsy material a "necrotizing involvement of the bridging veins which had allowed their contents to escape through their damaged walls." But as their case was also suffering from purulent meningitis the transudation theory previously mentioned might still conceivably have accounted for the effusion observed.

In Case 24 no such explanation is possible because there was no evidence of meningitis. The objection that the effusion was small can be answered in two ways. On the one hand, the thrombosis was so massive that the infant died before the effusion could grow to any size. On the other, there is a case in the literature (Penfield, 1923) in which a boy of 16 months was found to have huge bilateral subdural effusions after a febrile illness with otitis media. Penfield observed this boy for many weeks and noted further febrile episodes accompanied by a lymphocytic reaction in the cerebrospinal fluid. At that time little was known of the cause of "otitic hydrocephalus," as it was then termed, but Penfield obviously leans to this diagnosis in his discussion. The case would probably be regarded now as one of cerebral thrombophlebitis, and there is therefore no reason to regard Case 24 as unique.

Accordingly it may be concluded that cerebral thrombophlebitis, whether it occurs from extension of infection from purulent meningitis or from a focus elsewhere, may give rise to a subdural effusion, and that the likely mechanism is the giving way of the necrotic walls of veins crossing the subdural space.

Finally, now that we know (Symonds, 1952) how commonly an intracranial venous thrombosis may complicate any infective focus, however trivial and wherever situated in the body, it is tempting to attribute to the same process those cases of infantile subdural effusion in which convincing evidence of either trauma or intracranial suppuration is entirely absent.

Summary

A series of 18 cases of subdural haematoma of infancy is presented. The clinical features of the condition are described and the method of treatment is indicated. There is reason to suppose that nearly 70% of these children will grow up normally.

Five cases of subdural effusion complicating bacterial meningitis are described. The clinical details are given, and the similarity of the effusion to that found in subdural haematoma is noted.

A case of subdural effusion complicating sagittal sinus thrombosis is also described. On the basis of this case and of those of other workers it is concluded that certain types of subdural effusion complicating purulent meningitis may be due to a leakage of blood from the necrotic wall of a vein involved in thrombophlebitis.

It is a pleasure to acknowledge the co-operation of all the paediatricians in the Manchester Region in collecting the material for this paper. I am also indebted to Mr. L. P. Lassman and Mr. A. Jolleys, and to many residents at the Royal Manchester Children's Hospital, for help in the documentation and management of the cases, and to Sister N. Kewley for devoted nursing care. There is no doubt that the credit for the low operative mortality recorded in this paper belongs very largely to Dr. Tom Dinsdale, who gave the anaesthetics for almost all the operations.

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THE TREATMENT OF STEATORRHOEA IN CROHN'S DISEASE

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During recent years it has been recognized that Crohn's disease, or regional ileitis, is not always confined to the terminal ileum, but may be widespread, involving the proximal ileum, jejunum, colon, and even the duodenum. Surgery is definitely indicated for the relief of intestinal obstruction, and possibly for treatment of localized forms of the disease, when the affected portion of bowel may be excised. Patients with widespread disease present a difficult therapeutic problem. Surgery has proved disappointing, as complete extirpation of the affected bowel is seldom possible and recurrence after operation is common. In such circumstances therapy is directed towards improving the general nutrition of the patient and correcting deficiencies of vitamins and minerals. The main purpose of this paper is to describe the effects of a high-protein diet and intravenous iron, observed in three patients suffering from Crohn's disease with steatorrhoea, and to draw attention to the beneficial result that may accrue from a carefully planned diet.

Case 1

A schoolboy aged 12 was admitted to hospital on April 4, 1948, complaining of diarrhoea and of colicky pain in the right iliac fossa. Intermittent slight pyrexia was present, but otherwise his general condition was good. He passed several loose green stools daily, from which no pathogenic organisms were isolated. Agglutination reactions to the typhoid and dysentery groups were negative. A barium enema revealed nothing abnormal. After treatment with sulphonamides and penicillin he was discharged home on May 27. His symptoms persisted and his weight fell from 7 st. 3 lb. (45.8 kg.) to 4 st. 10½ lb. (30.2 kg.). He was readmitted to hospital on July 2. Apart from his wasted appearance clinical examination was negative. A hypochromic anaemia was present: Hb, 13.6 g. per 100 ml.; red cells, 4,300,000 per c.mm. The E.S.R. was 28 mm. in one hour (Westergren). Agglutination reactions were again negative, including those to *Brucella abortus*. The diagnosis of Crohn's disease was considered at this time, but attempts to demonstrate the terminal ileum radiologically failed. A low-residue diet was prescribed and his weight had risen to 5 st. 1 lb. (32.2 kg.) when he returned home on August 19. He remained under observation until March, 1949, when he weighed 6 st. 11½ lb. (43.3 kg.), and although his stools were still loose he was free from pain and attending school regularly.

In September the diarrhoea became worse and colicky abdominal pain returned, accompanied by vomiting. In April, 1950, examination by barium enema and sigmoidoscopy was normal. His condition deteriorated steadily until he was admitted to hospital on February 7, 1951, weighing 4 st. 3 lb. (26.8 kg.). He was passing about 10 loose offensive stools daily and vomiting most of his food shortly after it was eaten. He also complained of numbness and paraesthesiae in the extremities. Clinical examination revealed gross emaciation and faint brown pigmentation of the skin. The blood pressure was 110/75. No mass could be felt in the abdomen.

The results of laboratory investigations at this time are summarized briefly: E.S.R., 40 mm. in one hour (Westergren); Hb, 63% (9.3 g. per 100 ml.); red cells, 3,350,000 per c.mm.; C.I., 0.95; M.C.D., 7.2 μ; white cells, 12,000 per c.mm. The iliac marrow showed a marked normoblastic reaction of iron-deficiency type; macronormoblasts were also present, but no megaloblasts were seen. Microscopy of the stools showed no exudate; a few undigested meat fibres were seen. No faecal pathogens were isolated. Blood culture was negative. The titre of serum agglutinating *Br. abortus* was 1:640 ± 1:1,280, and *Br. melitensis* 1:320 ± 1:640. The total serum proteins were 5.4 g. per 100 ml. (albumin 3 g., globulin 2.4 g.). The average daily output of fat in the faeces was 7 g.; full details are given in the Table.

Radiological examination of the bowel showed marked narrowing and irregularity of the terminal ileum. There was narrowing in the middle of the transverse colon, with destruction of normal mucosal pattern, continuing to halfway down the descending colon.

As a result of the above findings a clinical diagnosis of chronic ileocolitis due to Crohn's disease was made.

The temperature varied between 99 and 100° F. (37.2 and 37.8° C.) for the first six weeks in hospital. The agglutination reactions to *Br. abortus* and *Br. melitensis* were still at the same high titre a week after admission. Blood cultures for these organisms were negative. A course of aureomycin was given, 60 g. in 21 days. There was little effect on the temperature, but the titres fell to 1:80 to both organisms four days after starting the drug and to 1:20 one month later, and have remained at this level. In view of the rapid fall in titre of agglutinins, it was unlikely that active brucellosis was present.

Feeding presented a great problem at first, owing to vomiting and anorexia. The patient was given a low-residue, high-protein, low-fat diet, containing daily two or three eggs, liberal helpings of meat and fish, and 75 g. of protein in a proprietary preparation. Bread-and-butter, small quantities of sieved vegetables, and boiled potatoes were included. His condition improved rapidly, and at the end of three weeks his weight was 5 st. 5 lb. (34 kg.), a gain of 16 lb. (7.3 kg.). The hypochromic anaemia responded to iron, 30 ml. of "ferrivenin" being given intravenously in the week before he was discharged from hospital.